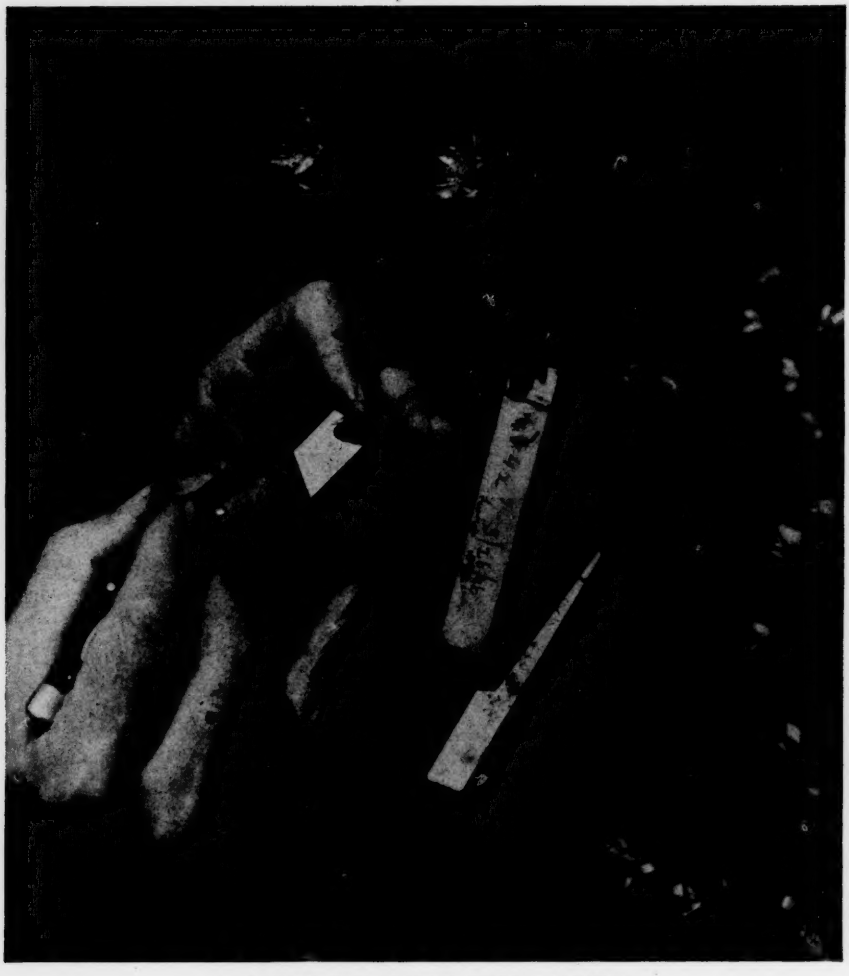
The Journal of HEREDITY



A SCIENTIST SEEKS THE ANTHER

(SEE PAGE 78)

Four-Footed Chickens

Heredity and Mongolian Idiocy

VOLUME 38 MARCH, 1947 NUMBER 3

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

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The JOURNAL OF HEREDITY is published monthly by the AMERICAN GENETIC ASSOCIATION at 32nd Street and Elm Avenue, Baltimore, Md., and 1507 M St., N. W., Washington, D. C. Address all communications regarding editorial matters to 1507 M. St., N. W., Washington, D. C. Communications regarding the business management of THE JOURNAL OF HEREDITY may be addressed to the publication office, 32nd Street and Elm Avenue, Baltimore, Md., or to 1507 M St., N. W., Washington 5, D. C. Entered as second-class matter February 24, 1923, at the postoffice at Baltimore, Maryland. Acceptance for mailing at the special rate of postage provided for in section 1103. Act of October 3, 1917, authorized December 29, 1918.

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The
Journal of Heredity

Vol. XXXVIII MARCH, 1947 No. 3

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Genetics, Medicine and Man

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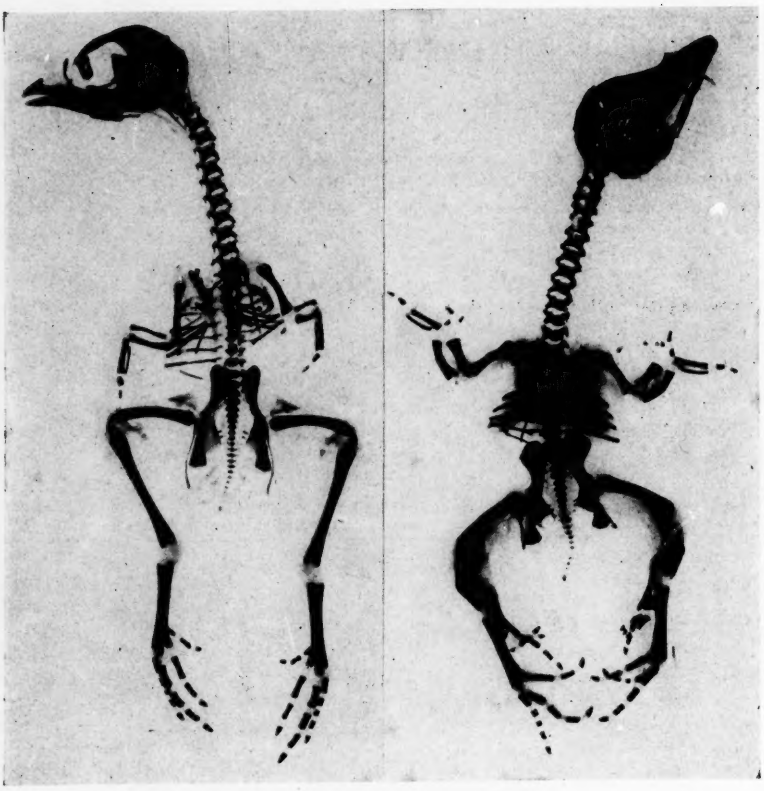


NORMAL AND "EXTRA-FOOT" CHICKS

Figure 1

Alizarin-stained, 17-day embryos from carrier dam A1161. *At left*—normal chick, except for absence of the rudimentary digit at the base of the metacarpals on both wings. *At right*—diplopod chick exhibiting four toes on right leg and three toes on left in extra complement, four digits on right wing and two on left in position of the normal rudimentary digit.

DIPLOPODIA: A LETHAL FORM OF POLYDACTYLY IN CHICKENS

LEWIS W. TAYLOR AND C. A. GUNNS
University of California, Berkeley

SEVERAL lots of unhatched S. C. White Leghorn eggs were received from a California hatchery during the summer of 1941 for examination of causes of unsatisfactory hatching results. In one lot of fully incubated, unhatched eggs, originating from a commercial hatching-egg flock mated to males from pedigreed stock, three embryos showed the lethal mutation described in this report.

Upon recommendation, a routine examination of unhatched pedigreed eggs was instituted for the remainder of the hatching season at the breeding farm from which the males mated in the hatchery flock were obtained. In all, seven embryos out of 22 produced from eggs laid by two hens mated to the same male were identified as polydactyls. An arrangement was made to secure hatching eggs from these hens in the breeding season of 1942, as well as from any daughters reared from these dams in 1941, all to be mated with the male siring the polydactyl chicks.

During four weeks of the 1942 breeding season all hatching eggs from the two hens and from seven daughters were incubated at the Division of Poultry Husbandry, University of California, Berkeley. Polydactyl embryos were again obtained from the two dams originally identified as carriers of the mutation, and also from four of the seven daughters. In addition eggs were received from two other hens mated to another male which were found to produce this deformity. A total of 26 out of 111 embryos (23.4 percent) from these eight hens were polydactyl (Table I), suggesting that the lethal condition was produced by the homozygous recessive state of a single gene.*

Morphology of Diplopodia

The most common or typical expression of this new type of polydactyly (over 75 percent of cases) consists of six toes per leg being grouped in two complements of three toes each (Figure 2). Three toes of one complement are in the position of digits II, III and IV of the normal foot. The other complement, located on the medial side of the normal tarsometatarsus, represents the supernumerary group. The hallux and its related metatarsal bone are absent. The normal digits are of usual size for the chick, while the supernumerary digits are generally smaller and highly variable in size and in structure. The innermost (medial) toe of the supernumeraries is uniformly longer than the other two; the middle toe is the most variable in size, frequently being only a rudiment.

The relationships of the supernumerary digits to the bones of the normal shanks are best studied in alizarin-stained preparations (Figures 1 and 3). The shank of the normal chick contains three bones fused to form the metatarsus, at the distal end of which digits II, III and IV are articulated. Digit I of the normal foot, the hallux, is attached by its metatarsal to the posterior side of the metatarsus at a point above the articulations with the other toes.

In the polydactyls illustrated in Figures 1 and 3, there are supernumerary metatarsal bones as well as extra digits. In many of these cases, each digit articulates with its corresponding meta-

*Recently Dr. W. Landauer of the Connecticut Experiment Station has informed us of his ability to produce phenocopies of diplopodia by insulin treatment of chick embryos in early incubation stages.

tarsal. The number of supernumerary metatarsals, however, is extremely variable, ranging from one to three or four. What appears to be distal branching of supernumerary metatarsals is common (Figure 3, right); not infrequently, points of articulation of three or four toes may arise from the branched ends of one or two metatarsals. The fact that, in typical expressions of this character, supernumerary metatarsals are constantly present, differentiates this form of polydactyly from others previously described in the chicken and has led to the adoption of the term *diplopodia* as descriptive of this condition involving doubling of the structures of the foot.

While the most common expression of diplopodia has been given above, it must be emphasized that numerous variant forms occur. These forms exhibit a variable number of supernumerary digits in the extra complements associated with occasional absence of supernumerary metatarsals and the presence of the normal hallux. Unilaterally the extra complement in about 10 percent of the diplopods is composed of four toes (Figure 3, right), the extra digit apparently arising as a partial or complete duplication of the middle toe of the usual supernumerary complement of three. In approximately an additional 10 percent of the diplopods there is either a unilaterally or bilaterally expressed lesser number of toes than the usual three in the extra complement. By far the most frequent types found here are those involving only two toes in the supernumerary complement. In the remaining cases (about 3 percent of all diplopods), the normal hallux is retained and the extra complement consists of from one to three toes (Figure 4). The presence of less than three toes in the extra complement has been considered as an atypical expression of diplopodia.

As indicated above, asymmetrical development in number of toes in the extra complement is common. In a representative sample of over a hundred diplopod chicks, 16.5 percent showed asymmetry in number of toes. Of these, in five-sixths of the cases the right leg possessed more toes than the left. An exceptional case is shown in Figure 4. The asymmetry in diplopodia is a complete reversal of the condition in common polydactyly, where Bond[1] reported only four out of 38 heterodactyls had the extra toe on the right side.

Sex in diplopod embryos closely approaches the expected 1:1 ratio of males to females. No variations in expression of the mutant gene seem to bear any relation to the sex of the embryo; all evidence points to the conclusion that the gene is borne on an autosome.

Other Morphological Effects of Diplopodia

Shortening of the femur, tibia and tarsometatarsus occurs in all typical diplopod embryos, the most severe reduction in length being found in the tibia (Figure 1, right). Frequently, pronounced dystrophies of these bones, particularly of the tibiae, occur, leading to an abnormal curvature of the legs. As a result, diplopod embryos often assume a hatching position in which the legs are placed posterior, instead of ventrolaterally, to the abdomen. In atypical expressions of diplopodia, the length of the leg bones is usually intermediate between that of the typical diplopod and the normal embryo. No duplication of structures of the leg above the tibiotarsometatarsal joint has been observed.

Supernumerary structures in the wing are associated with typical diplopodia in approximately 97 percent of the chicks. In the normal chick there is a rudimentary digit near the base of the larger of the two metacarpal bones of the wing (Figure 5, upper). This has been interpreted variously as a pollex (digit I) or as digit II; a recent embryological study[8] favors the latter view. At the normal position of this digit, diplopod embryos usually show complexes of from two to as many as four digits (Figures 5, lower, and 6). Usually the digits have but a single phalanx; occasionally that digit nearest the adjacent normal metacarpal

FOUR-FOOTED CHICKS
Figure 2

Diplopod embryos at right and left; normal chick in center. Diplopod at left exhibits short maxilla.

has two phalanges. In the most extreme development of these supernumerary structures, bones identifiable as metacarpals may be found. The radius, the ulna and the metacarpals are shorter than normal. The wing of typical diplopod embryos, therefore, shows a tendency towards duplication of structures and shortening of bones paralleling that found in the leg.

A shortened maxilla is found in over 80 percent of the cases of diplopodia, and very rarely in normal embryos from the same matings. This condition is illustrated by the embryos in Figures 2, left, and 6.

Lethality of Diplopodia

Only rarely do diplopod chicks hatch. Of 423 diplopods produced in the five years of this study, only nine or 2.13 percent have hatched. Handicaps arising at least in part from the shortening and curvature of leg bones and from the commonly associated defect of the upper beak apparently cause death of many fully developed embryos at terminal stages of incubation. Death of recognizable diplopod embryos at earlier stages in incubation is at a rate comparable to the loss of embryos not exhibiting this deformity. The fact that the proportion of diplopods identified from carrier × carrier matings in four of the five years fell short of the expected 25 percent level (Table I) may suggest a higher death rate of these embryos during the first week of incubation, when the mutation can not be identified with certainty. An alternative explanation might be that a small proportion of genetic diplopods are phenotypically indistinguishable from normal chicks.

Of the nine diplopod chicks hatched, all save one, an atypical diplopod, died within a few days. This exceptional chick, a female, had one supernumerary plus four normal toes on each leg. This bird was raised to maturity, but was abnormally small in size and laid only

two shell-less eggs before death. The typical expression of diplopodia is a completely lethal condition, since even under most favorable brooding conditions the chicks do not survive.

Hereditary Nature of Diplopodia

Beginning with 1942, stock for matings was saved mainly from carrier × carrier matings. In 1943, these birds were tested in a series of matings. Of eleven females tested, nine proved to be carriers, while of ten males used seven were carriers. Since two-thirds of the normal chicks from carrier × carrier matings would be expected to be carriers themselves, if diplopodia were produced by the recessive condition of a single gene, the actual number of carriers, 16, slightly exceeded the expected number, 14.

The stock from the poultry breeding farm proved to be extremely susceptible to lymphomatosis. In order to save the mutant gene for further study, it became necessary to outcross a carrier male by mating with females from a non-related, more resistant line from the University White Leghorn flock. This was done late in 1943.

In 1944, six of the F_1 females reared from the outcross were tested by mating with known carrier males from the original source; two females proved to be carriers. Offspring hatched from both carrier and non-carrier dams were reared.

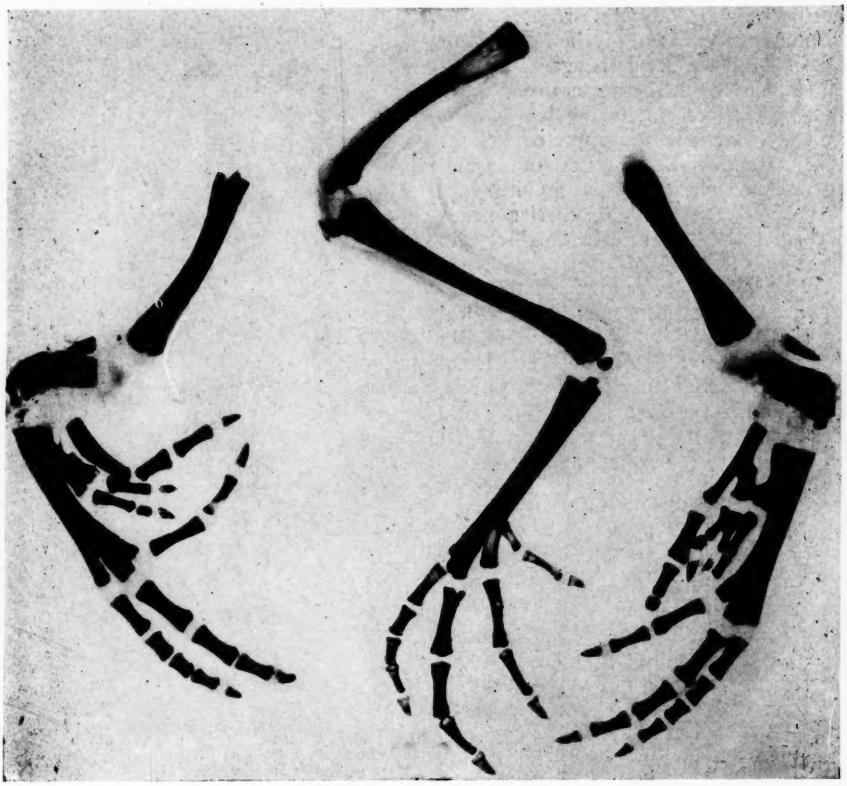
Of four females from the carrier × carrier matings of 1944, one was proved to be a carrier by tests made in 1945. Four males from these matings were also tested; only one proved to be a carrier. Instead of an approximation of the expected 2:1 ratio of carriers to non-carriers, a distribution of 2:6 was actually obtained for all offspring. This represented an unexpected deficiency of carriers.

Of twelve females derived from carrier × non-carrier matings, three were proved to be typical carriers, two produced abnormally low ratios of diplopod embryos when mated with carrier males, and seven were non-carriers. The two females producing the unexpectedly low proportion of deformed embryos were full sisters. Instead of the expected 25 percent of diplopod embryos from carrier × carrier matings, only approximately 11 percent were obtained, and this reduction in ratio of defective embryos held in one bird for matings with three different carrier males. The actual normal to diplopod distributions in

TABLE I.—Summary of Mating Results by Years

Year	Mating* ♂ × ♀	Normal	Diplopod	Percent Diplopod
	Carrier × Carrier			
1942	B×B	85	26	23.4
1943	B×B	237	83	25.9
1944	B×BC	48	13	21.3
1945	B×(B×BC) or (B×BC)×(B×BC)	299	89	22.9
1946	(B×BC)×(B×BC) or (B×BC)×[B×(B×BC)]	514	137	21.0
All		1183	348	22.7
	Carrier × Non-Carrier			
1942	B×B	29	0	0.0
1943	B×B	51	0	0.0
1943	B×C	14	0	0.0
1944	B×BC	141	0	0.0
1945	B×(B×BC)	247	0	0.0
1946	(B×BC)×[B×(B×BC)]	251	1	0.4
All		733	1	0.14
	Carrier × Atypical Carrier			
1945	B×(B×BC) or (B×BC)×(B×BC)	205	25	10.9
1946	(B×BC)×(B×BC) or (B×BC)×[B×(B×BC)]	214	41	16.1
All		419	66	13.6
	Non-Carrier × Carrier			
1943	B×B	36	0	0.0
1944	B×(B×BC)	13	0	0.0
1945	(B×BC)×(B×BC)	81	8	9.0
1946	(B×BC)×(B×BC) or (B×BC)×[B×(B×BC)]	367	0	0.0
All		497	8	1.6
	Non-Carrier × Non-Carrier			
1943	B×B	3	0	0.0
1944	B×BC	47	0	0.0
1945	(B×BC)×(B×BC)	102	0	0.0
1946	(B×BC)×[B×(B×BC)]	58	0	0.0
All		210	0	0.0
	Non-Carrier × Atypical Carrier			
1945	(B×BC)×(B×BC)	60	0	0.0
1946	(B×BC)×(B×BC) or (B×BC)×[B×(B×BC)]	180	0	0.0
All		240	0	0.0
	Non-Carrier × Unknown Constitution			
1946	(B×BC)×[B×(B×BC)]	24	0	0.0

*B = from breeding farm, C = from University.



DETAILS OF DIPLOPODIA
Figure 3

Alizarin-stained legs of two diplopod embryos and of one normal. The diplopod leg on the left shows three extra metatarsals and a complement of three extra digits. The leg at the right possesses two metatarsals (one branched distally) and four digits in the extra complement.

1945 were for female A1151—58:8, 2:0 and 1:0 and for female A1156—90:12, 10:1 and 43:5. The significance of the deviations of these data from the expected 3:1 ratios may be interpreted from the $\chi^2 = 22.4$ with $P = .001$ approximately. These dams also tended to produce a higher proportion of chicks exhibiting atypical expressions of diplopodia, the incidence being 20 percent or double that from typical carrier dams. Since half of the twelve daughters of the carrier × non-carrier mating would be expected to be carriers themselves and only three typical carriers were identified, the two atypicals may be considered to be carriers which have in some way been inhibited in the production of the usual ratio of diplopod chicks when mated to carrier males.

In 1946, three carrier females and the two atypical carriers were used in matings along with 21 pullets derived from four carrier × carrier matings of 1945. Of the pullets, eleven proved to be carriers, three atypical carriers, five non-carriers, and two died before proper matings could be made to test their

genotype. The atypical carriers were produced from two of the four 1945 matings. If the atypicals again are classified as carriers, there was a slight excess of carriers among the fully tested pullets (14 against an expected 12.67). The individual distributions of normal to diplopod chicks for the atypical carrier pullets mated with carrier males were: B1607—34:4; B1616—34:4 and 28:4.

Of the atypical carriers of the previous year, one performed like a typical carrier in 1946 (A1151—31:13 and 21:7), while the other remained consistent in the low proportion of diplopods produced (A1156—36:6 and 28:3). These results gave a two-year total for A1151 of 113:28 or 19.9 percent and for A1156 of 209:27 or 11.4 percent diplopods. Thus despite the apparent significance of the very low number of diplopods from the first mating of A1151 in 1945, reasonable doubt may be cast upon the accuracy of the classification of this bird as an atypical carrier. On the other hand, A1156 produced a consistently low proportion of diplopod chicks in matings with five different carrier males and B1616 also did so with two males. Some doubt may be expressed, therefore, concerning the accuracy of classification of a female as an atypical carrier from results of but one mating. In Table I the inclusion of A1151 in the atypical carrier female totals raised the percentage of diplopods with carrier mates to 16.1 in 1946 whereas if she were excluded the percentage would be 11.5, as compared with 11.2 for A1156 alone in 1945. Both of the latter percentages are approximately half the average percentage of diplopods obtained (22.7) by mating typical carriers together.

Sufficient study of the breeding performance of offspring from atypical carrier dams has not yet been made to determine whether the basis of partial inhibition of the production of diplopods is genetically or environmentally determined. The appearance of atypical carrier females after only one outcross of the original stock to the University strain and the close relationship existing between such females favor the interpretation that one or more genetic inhibitors have been derived from the University strain.

It should also be noted (Table I) that in 1945 eight diplopods were obtained from a mating of non-carrier males × carrier females, or an incidence of 9 percent. No certain explanation can now be offered for this unexpected result.

AN ATYPICAL DIPLOPOD EMBRYO
Figure 4

This individual has normal wings, asymmetrical development of the toes and absence of supernumerary metatarsals. Each leg has four normal toes; the right extra complement has one, the left has three toes. This case is exceptional since most asymmetrical cases have the greater number of toes on the right leg.

EXTRA "WING-FINGERS"
Figure 5

Alizarin-stained wings of normal and diplopod embryos. The diplopod wing has four digits and three metacarpals in the extra complement.

These eight embryos were obtained from two matings: one from A112 male × A1149 female out of 21 embryos, the other seven from A111 male × A1161 female out of 57 embryos. Both dams gave normal ratios of diplopods when mated to carrier males. Unfortunately these males were not mated to other carrier dams, thus it is not known whether these males were atypical carriers in the same sense as the atypical dam A1156. A112 male was mated to both A1151 and A1156 during this year but gave no diplopods in a total of 47 chicks. In 1946, one diplopod was produced from a mating of a carrier male × non-carrier female; this might have been a case of error in the identification of an egg in trapnesting.

Comparison of Diplopods with Other Polydactyls

The most common form of polydactyly in fowls is that represented in a number of breeds possessing five toes per foot. This form is produced by an incompletely dominant gene P_o and is characterized by a fifth toe situated close to the hallux. Asymmetrical development of the fifth toe is common, the extra digit appearing more often on the left leg only,[1,2] Modifying factors have been postulated as responsible for the occasional appearance of four-toed birds which are proved carriers of P_o by breeding tests.[4,6] A modification of the common type of polydactyly, called polyphalangy by Warren,[11] results in the production of four toes per foot, but digit I becomes relatively long and possesses an extra phalanx. Fisher[4] also noted an increased number of phalanges in the halluces of birds from polydactyl stock. Sturkie[9] has produced evidence that incubation temperatures are an important factor in the suppression of polydactyly. Diplopodia differs from common polydactyly in many respects, such as sidedness of asymmetry, associated deficiencies of the maxilla, viability of the chick,

FOUR-HANDED CHICK

Figure 6

Diplopod embryo showing unusually complex structure of the extra wing complements. Dissection of this specimen revealed well-developed supernumerary metacarpals and digits with as many as two phalanges.

and its determination by a recessive gene, as well as in the fundamental structure of the extra digits.

Warren[10] has described another form of polydactyly, termed "duplicate," which occurred in White Leghorn stock. Duplicate chickens showed apparent doubling of the foot in extreme cases, but there was great variability in expression of the defect. The usual form is represented by an extra toe added just beside the hallux. Duplicate also behaved as an autosomal dominant; later Warren[10] obtained evidence that the gene for duplicate is located at the same locus as P_o and is a member of a series of multiple alleles. Duplicate, like common polydactyly, is subject to suppression by both environmental and genetic factors.

A condition known as "talpid," a lethal factor usually producing death in embryos at from eight to ten days of incubation, is also characterized by duplication of toes and digits of the wing.[3] This condition was proved to be a simple autosomal recessive. Talpid embryos show very poor differentiation of the digits of the feet and wings. A 17-day embryo figured by Cole shows extreme reduction in limb size and the limbs do not appear to indicate a duplication of the feet or of the rudimentary hand structures in the wing. The talpid mutation usually expresses its lethal action earlier than diplopodia. It does not appear likely that diplopods are talpids with a lesser reduction in size of wing and leg and with greater viability.

Diplopodia differs morphologically from all of the above forms of polydactyly. Genetically it is like the talpid mutation in that both are autosomal recessives. Morphologically the closest approximation to the typical diplopod embryo is found in the most complex cases of duplicate; a few atypical diplopods are similar in appearance to other

extreme variants of duplicate. Warren's description of duplicate does not include information on the number of metatarsals present. After examining a typical diplopod chick, he has expressed the opinion to us that the mutation is not identical with duplicate.

Diplopodia may possibly represent a reduplication of the feet. In such cases the supernumerary structure would be expected to develop as a mirror-image of the normal metatarsus and toes. A study of the number of phalanges in the digits of the extra complements in a limited number of alizarin-stained, diplopod embryos has given some imperfect resemblance of reduplication in about half of the legs examined; in the remaining legs there was only one case where the evidence has appeared to be distinctly against reduplication. At present it is not possible to state definitely whether or not diplopodia is an incomplete form of reduplication.

Gabriel's recent paper[5] on the effect of local applications of colchicine on chick embryos is of special significance in considering the nature of diplopodia. His case of a six-toed embryo derived from an experimentally treated blastoderm of the common form of polydactylous stock (*cf.* his Figure 2) involves duplication of the "cannon-bone" or three fused metatarsals and mirror-imaging or reduplication of digits. This embryo shows many features in common with diplopods in the structure of the supernumerary complements. Gabriel's hypothesis that polydactylism is not the result of added halluces but the partially inhibited expression of a duplicated limb field finds support in the production by natural means of the closely similar mutant condition described in this paper and termed diplopodia.

Explanation of the Poor Hatchability in the Breeding Flock

The three diplopod embryos obtained from unhatched eggs from the commercial breeding flock represents an incidence of 0.36 percent in terms of the original number of fertile eggs in the lot. The hereditary lethal condition originating from this source was obviously only a minor factor in determining the excessive rate of embryonic mortality, but, on the assumption that 0.36 per cent represents q^2 in the Hardy-Weinberg binomial $(p+q)^2$, it may be concluded that the concentration of the recessive allele in the hatchery-flock population was 6 percent, and that the percentage of carriers of the recessive gene in that flock was $\frac{2pq}{p^2+2pq}$, or 11.3 percent. This represents a high level of dissemination of the lethal gene in the flock. In view of the fact that the hatchery flock was of relatively large size and that there is no *a priori* reason to believe that the carriers were favored in selection, this incidence may be interpreted to indicate that the mutation must have originated a number of generations before its discovery.

In studies made at other poultry farms, it has since been determined[7] that poor egg quality often resulted as an aftermath of the disease pneumoencephalitis. Since this disease had occurred in the hatchery flock previous to the appearance of poor hatching results and since the eggs showed defective shells and membranes of the type later described by Lorenz and Newlon, it was probably the major cause of the unsatisfactory hatchings.

Summary

A condition termed diplopodia is described which differs from all known forms of polydactyly in chickens. The mutant is characterized by partial doubling, possibly a reduplication, of the metatarsals and digits of the feet and of the metacarpals and digits of the wing. Usually associated with this condition is a shortening and curvature of the long bones of the leg and wing and also shortening of the maxilla. It is inherited as a simple autosomal recessive, although there is evidence of a suppression of the action of the gene in some cases by either environmental or

genetic factors. It is lethal to the extent that approximately 98 percent of the diplopod chicks die in the shell. Of nine diplopods hatched only one atypical individual has been reared and this proved so abnormal that reproduction from it was not possible. Diplopodia differs from other forms of polydactyly in the chicken genetically, morphologically, in the expression of asymmetries, in associated deficiencies of the maxilla, and in the expression of its lethal nature. It does, however, closely resemble a form of polydactyly in a chick produced by experimental treatment of a polydactylous blastoderm by Gabriel.[5]

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BLINDNESS AND HEREDITY

THE Public Affairs Committee covers a lot of ground in its pamphlet series. Such diverse subjects as the 59¢ dollar, epilepsy, cartels, race problems, aviation, and the radio have been discussed. A recent addition to the series* is titled *What Do You Know About Blindness?* In general, its discussion of the problems of the blind and of people who deal with them appears to be excellent. The causes of blindness are discussed in some detail. Gonorrheal conjunctivitis used to be responsible for a fourth of all blindness; it is now down to six per cent thanks to prompt use of silver nitrate drops in the eyes of an overwhelming majority of babies. The fact that one blind person in sixteen still owes his affliction to a neglect of this easy preventative shows how hard it is to close even an obvious gap between knowledge and practice.

Syphilis causes 15 percent of blindness, and trachoma remains an important factor. Cataract is today the most frequent cause of loss of sight. In a sample of 20,000 blind adults, one fourth of them were blind for this reason. Among a large group of blind children, 15 percent were blind because of congenital cataract. Glaucoma contributes 11 per cent of the cases of blindness among middle-aged and older persons. Accidents are responsible for 15 percent of blindness. Fifteen hundred American soldiers and sailors lost their sight during World War II.

This discussion is followed by a brief statement concerning "Heredity's Part" in the causes of blindness. It is recognized that inherited eye defects play an important part in the problem of the blind, but the author underestimates the contribution of heredity because of the following peculiar misconception (the italics are the reviewer's):

> Heredity, however, does not play quite so large a part in blindness as was once supposed. The Committee on Statistics of the blind has compiled figures on the causes of blindness

(*Continued on page* 88)

*Yahres, Herbert. *What Do You Know About Blindness?* Pp. 32. 10¢. Public Affairs Committee, 22 E. 38th St. New York. 1947.

INDUCING POLYPLOIDY IN PEACH VARIETIES

HAIG DERMEN*

IN mid-April of 1944, five 10-year-old Elberta peach trees in the orchard were cut back to about five main branches to obtain vigorous new shoot growth for colchicine treatment. By May 5, when the colchicine treatment was made, these trees had produced new shoots, varying in number from one to ten, in stages of growth from barely-emerging to five centimeters long. Perhaps because of the severity and lateness of pruning, the weakness of some of the trees, and the very young stage at which some shoots were treated, three of the five trees died. All shoot tips were trimmed back close to the terminal growing point in preparation for treatment. A one-percent solution of colchicine in water was used, to each 10 cc. of which were added two drops of 10 percent Santomerse wetting reagent. The amount of solution applied to the tips (by means of a medicine dropper) was one to two drops, only enough to wet the treated parts thoroughly. The treatment was repeated once a day for five consecutive days.

At the end of the first year there was no obvious indication that polyploidy had been induced in the terminal growth in any of the shoots, but a polyploidy effect was evident in a few leaves of the growing shoots at the treated area, from the fact that these leaves were distorted and portions of them were thick or showed enlargement of stomates. In 1945 on one tree a small branch was discovered, the leaves of which had large stomates, indicating tetraploidy, at least epidermally. The only available buds of this twig, five in number, were budded into seedling understocks, and three young trees resulted from this budding. The appearance of the leaves of these trees indicates that only the epidermis was affected; therefore fundamentally the plants are diploid. However, the effect, if any, of the epidermal polyploidy on the fruits and on the general physiology of the new plants will be watched with interest.

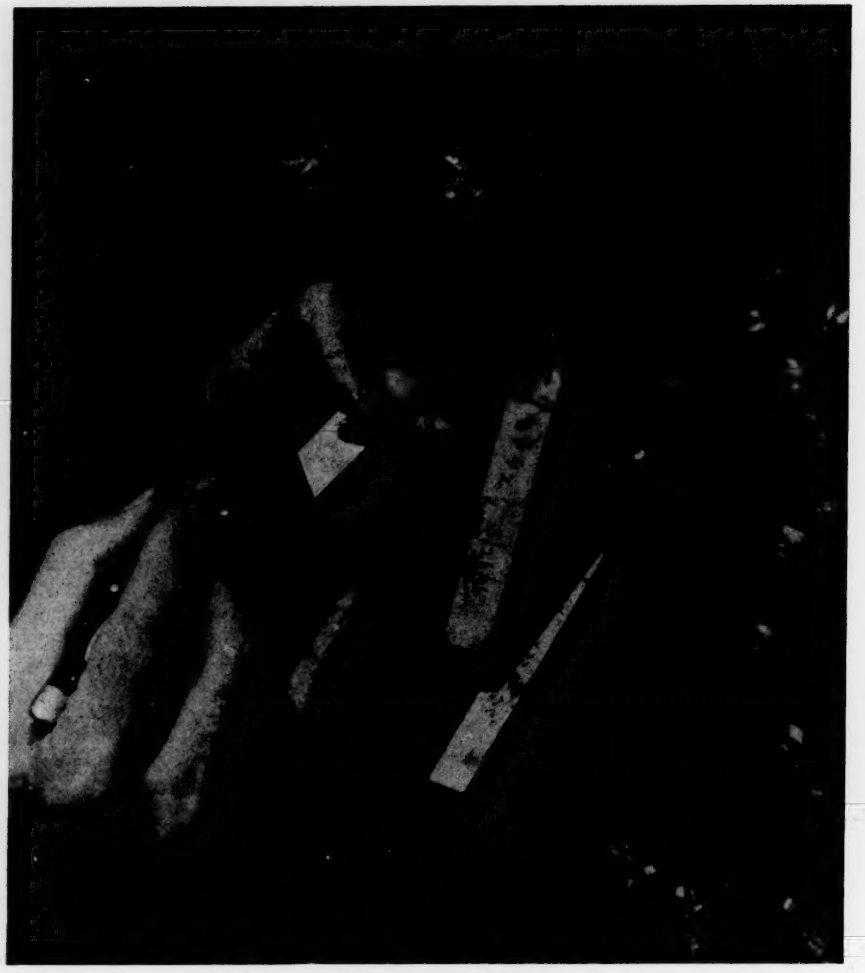
Internal Polyploidy Induced

On March 26, 1946, another branch was found in the vicinity of the branch just described, along one side of which six flowers seemed somewhat larger than the normal-appearing flowers on the other side of the branch (Figure 7). The anthers of the six flowers were strikingly large. Anther size has been found to be the best gross characteristic for selecting flowers suspected of being internally polyploid. Internal polyploidy in these flowers was then confirmed by pollen grain examination.

A few anthers from each flower were placed on separate slides smeared with a small amount of egg albumin preparation (Figure 7), and were allowed to dehisce on the slides in the laboratory for examination. Normal pollen grains (monoploid) are shown in Figure 8, upper. Figure 8, lower, shows large pollen grains (diploid) from one of the large flowers. In staining, most of the polyploid pollen grains appear four-sided, in contrast to the triangular appearance of normal pollen grains (Figure 8). About 25 percent of the pollen grains in one of the polyploid flowers were aborted; of the remainder, less than one percent were large, dyad grains (tetraploid), as shown in Figure 8. The pollen sample from the normal diploid flower contained about 10 percent of aborted grains.

From only one of the polyploid flowers a fruit began to develop, but it

*Associate Cytologist, Division of Fruit and Vegetable Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, United States Department of Agriculture.

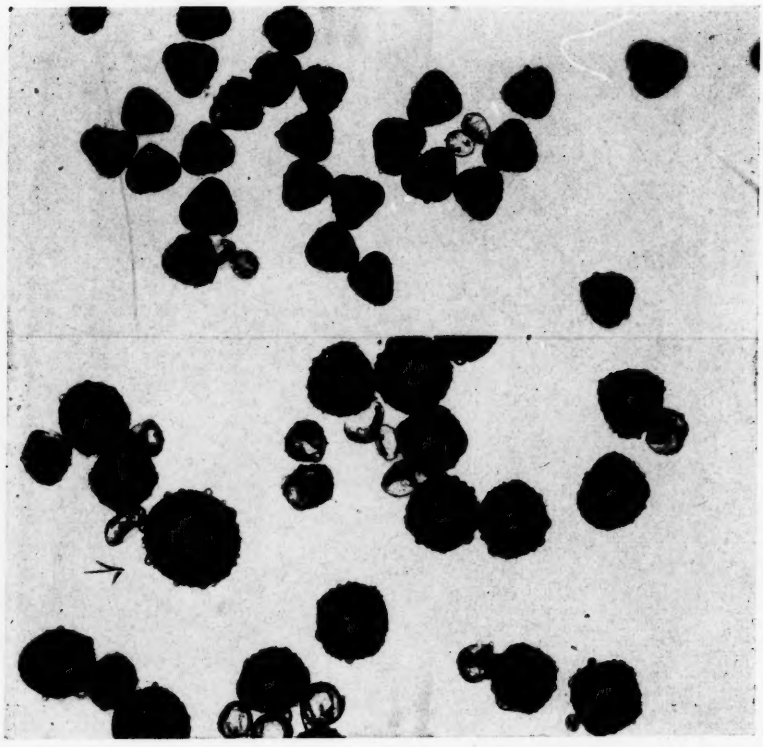


POLYPLOID FLOWER
Figure 7

Of the upper two flowers, the one on the left is normal and the one on the right polyploid. The operation in process is the gathering of anthers on a slide to determine through microscopic examination of the pollen the ploidy of the flowers.

dropped off after it had reached about one centimeter in diameter. It was noted that this fruit was definitely round, with a depressed end, in contrast to the normal elongated fruits, with pointed stylar ends, of the Elberta variety at the same stage of development.

At the end of the 1946 growing season only a short twig had developed on the polyploid side of the branch. The leaves on this twig were epidermally normal, but looked somewhat broader and thicker than normal. The twig had only three prominent buds, which were



NORMAL AND POLYPLOID POLLEN

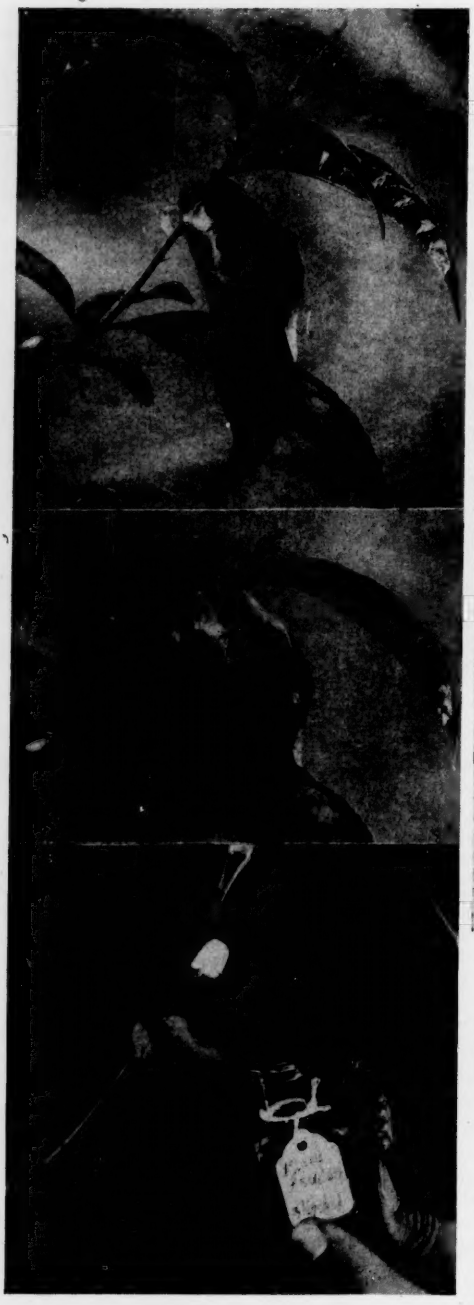
Figure 8

Stained pollen grains of the normal flower above appear triangular. Hyaline grains are aborted. They are transparent. About ten per cent of the pollen grains were aborted. Pollen grains of polyploid flowers below appear four-sided and are considerably larger than those of the normal flower. Twenty-five per cent of these grains were aborted. A dyad (tetraploid) grain is indicated by the arrow.

used for budding on understocks with the expectation that the resulting trees would be internally polyploid.

Polyploidy Under Glass

Polyploidy was also induced in greenhouse-grown peach trees. Nine Elberta and ten Golden Jubilee one-year-old peach trees were grown in sand culture in 3-gallon crocks, two trees to a crock, and in addition six Golden Jubilee one-year-old trees were grown in soil in 12-inch pots, one tree to a pot. These three lots of trees were planted February 28, March 18, and April 3, 1946, and colchicine was supplied March 22, April 12, and May 13, respectively. Since these trees were to be transplanted and grown in the orchard, it was necessary to have growth high enough above the ground to avoid injury to the branches from cultivation and to manage the growing branches. Therefore, shoot growth was stimulated two feet above the sand and soil level by cutting back the tree to a bud at that height. Only one vigorously growing shoot at the highest point of the stem was allowed to grow and all other growth was cut off. Treatments, as indicated above,

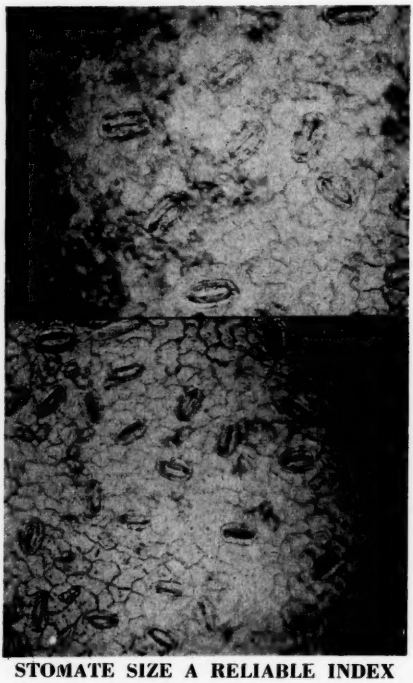


HALF-AND-HALF
Figure 9 (above)

A sectorially polyploid leaf. The left side is totally diploid; the right side is totally polyploid. Internal polyploidy rather than epidermal polyploidy is responsible for the difference in width of the two halves.

COLCHICINE TREATMENT
Figure 10 (at left)

A shoot at the stage at which colchicine was applied is shown in the top photograph. Below this is shown the tip of this shoot prepared for colchicine treatment. The final photograph shows an absorbent-cotton pledget in place over the shoot tip to keep it shaded and to keep it moistened with colchicine for a longer time. For treatment, see the text.



STOMATE SIZE A RELIABLE INDEX

Figure 11

Large stomates of a polyploid epidermis compared with the smaller stomates of a diploid epidermis. Positive identification of polyploid plants and sectors depends on chromosome counts in the shoot tips.

were made three or more weeks after planting the trees, when the shoots had made five or more centimeters of growth. In preparation for treatment the young leaves near and around the shoot tips were removed and the very young leaves and stipules at the very tips were cut off with a sharp blade to come as near as possible to the terminal growing point. In removing the leaves at the shoot tips, care was taken not to cut into the stem, to avoid injury to the bark of the young shoots. Such injuries cause wilting or killing of the tender tips. At times the cuts were accidentally made deeper, so that the terminal points were cut off; but these shoots were, nevertheless, used for the treatments. The lower leaves of the shoots were left to carry on the essential growth processes of the trees. Figure 10 shows the stages of a shoot tip prepared for the treatment. The tips were covered with a pledget of absorbent cotton, which served to absorb the colchicine solution and to keep the tips moist. The pledgets also, by shading the tips, prevented them from drying out.

One Elberta and one Golden Jubilee tree were left as controls. The shoot tips on the controls were similarly prepared, but wetted once a day with water until the end of repeated colchicine treatments. Four Elberta trees were treated with a solution consisting of 0.7 percent of colchicine in 37.5 percent of glycerine in water. To each 10 cc. of solution were added nine drops of 10 percent Santomerse. The four remaining trees were treated with one percent water colchicine solution with two or three drops of Santomerse. The glycerine-colchicine treatment was made only once, but after this treatment the tips were wetted with water once daily for 10 days. *All glycerine-treated tips abscised.* However, one newly developed lateral shoot below the glycerine-colchicine-treated tip on each of three of the four trees was used for water-colchicine treatment. Shoot development on one of the four trees being too slow, it was not used for the treatment. The water-colchicine treatment was repeated four times at three-day intervals. The tips were wetted with water once daily between the times of treatment and for three days after the final treatment. The treatment with water-colchicine resulted in polyploidy in all Elberta trees, either in the main terminal growth or in the growth from axillary buds.

Golden Jubilee trees grown in sand and soil were treated with water-colchicine solution only; some four times, as with Elberta, some twice, and some once. In all cases the treatments on Golden Jubilee were, in varying degrees, injurious. However, in some trees lateral shoots developed from the treated

areas, which showed polyploidy from all three lots—those treated four times, twice, and once. Polyploidy was induced in five of the fifteen Golden Jubilee trees.

In the above experiment the trees were kept growing until the end of August in a section of the greenhouse with ventilators wire-screened to keep out injurious insects, especially the oriental fruit moth, the larvae of which bore into the shoot tips and destroy them. About a week before the above date, the upper part of the sand in the crocks was replaced with soil. Then the plants were transferred to a screen house for further growth and hardening. In mid-October these plants were planted in the orchard.

Discussion of Cytohistology

The cytohistological and morphological phases of colchicine polyploidy found in the cranberry[1,2,3] are in general applicable to the peach. In the peach, as in the cranberry, colchicine treatment results mostly in sectorial and periclinal diploid-polyploid chimeral forms, which may be recognized by certain features of the leaves. Leaf stomates of peaches are uniform in size. When the undersides of diploid and tetraploid leaves are observed together under a lens of ×10 or higher magnification, large tetraploid stomates stand out quite sharply in contrast to the small, normal stomates. Polyploidy of the internal tissue of the leaf affects the width and the texture of the leaf. That is clearly seen, especially when one half of a leaf is internally normal and the other half is internally tetraploid. The leaf in Figure 9 shows such a case, except that the wide tetraploid half of that leaf had also a tetraploid epidermis. The epidermis of the normal narrow half was diploid. The contrast between the stomates of the two halves of the epidermis is shown in Figure 11. A special technique is required to isolate and propagate the polyploid sectors induced in most plants; this is fully discussed in the articles on cranberry already referred to.

A Suggested Technique

Evaluating the results so far obtained from various methods used for peach treatments, the following procedure is suggested as most likely to be sucessful: Plant one-year-old trees singly in soil in 12-inch pots between mid-January and early February. Cut the plants back to a bud two feet above the soil level. Allow one shoot to grow to a length of five inches or more and destroy other growth. Break off the young leaves at the tip and cut off the remaining very young leaves and stipules with a sharp blade close to the terminal growing point. Cover the tips with a pledget of absorbent cotton. Leave one plant as control. Use a one-percent water-colchicine solution, with two drops of 10-percent Santomerse to each 10 cc. of the solution. The third day after trimming the tips apply colchicine solution to the pledgets, soaking them thoroughly; application of colchicine immediately after trimming the tips may be one cause of injury from treatments. Wet the pledgets with water once two days after the treatment. Wet the control tip whenever the treated tips receive either colchicine or water. Destroy all growth on the shoots below the treated area of the tips. Allow laterals to grow from any axillary buds that have been covered with the pledgets and been wetted with colchicine. If leaves showing sectorial or entire polyploidy are found in the upper region of the new growth, the shoots should be cut to such a leaf, and new shoot growth should be forced at that point, either during that season or the following year. For further details on this point, see the articles cited below. Colchicine experiments may be more successful if treatments are made early in the season when the weather is cool.

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MORE WISHFUL THINKING ABOUT MONGOLIAN IMBECILITY

THE author of this book* attempts to unravel the cause of Mongolian Imbecility and to rid it of its mystery. Mongolism, he maintains, results from a pituitary deficiency. He discusses cretinism which is caused by a thyroid deficiency as a parallel case. The scheme of the book is outlined in the headings of the chapters, which deal with the historical aspects of each disease, physical characters and diagnosis, mental development, the nervous system, endocrine pathology, general pathology, osseous growth, X-ray studies, hematology and biochemistry, relation of mongolism to the maternal condition, prevention of mongolism and principles of treatment.

The reviewers will not discuss the clinical and laboratory data. This review will confine itself to that portion of the book dealing with genetics in which the readers of this JOURNAL will be more interested. Space cannot be devoted to a critical review even of all this section of the book. Much of it is of interest, even though some of the author's conclusions may not be justified by the data.

How Many is "Most"?

First, the reviewers deplore the tendency toward exaggeration of the author's impressions without due regard to the facts elicited from his material. Here is an example:

Benda states, "Although many a mongoloid child has ten to thirteen siblings, in no family with such a large progeny has multiple incidence of mongolism been found." His Table 39 however, shows that only 14 out of 255 mongoloids had as many as "10 to 13 sibs," an incidence of 5.5 per cent. This is neither "many" nor even a *high percentage* of mongoloids. The second part of the sentence is contradicted by the next sentence in his text. Here he states that "in another family, . . . 2 of 13 children were abnormal." Thus he encountered one family at least in which each of two mongoloids had 12 sibs.

Other examples of this exaggeration in the face of the data are these: "If we add that *most* [italics reviewers'] of these women previously had one or more abortions, and that in addition the pregnancy which terminated in the birth of a mongoloid was threatened by abortion, then we see that the mongoloid is born under conditions which may be recognized in advance." (p. 284.) But we find that in his Table 43, 7 of 75, or only 9.3 per cent, had had one or more abortions; and 11, or 14.6 per cent, had threatened abortion. Only six of 60, or 10 per cent had a history of both. Therefore his statement that mongoloids are born under circumstances which may be recognized in advance because *most* mothers give significant histories is not sustained by the data presented.

Moreover, the 75 mothers with these histories would appear to have been especially selected from the complete series of 253 mothers of mongoloids because they presented a special "constellation of factors present during pregnancy." Either Benda did not enquire as to these factors in the remaining 178 mothers, or they presented no significant histories. If the latter alternative be true, even the above percentages would be materially reduced; if the larger group was not queried concerning these special factors, why not?

"Most of the Monogoloids are very much desired children, and therefore, attempted abortion is not a significant item." (p. 286.) In 67 case histories which Benda gives, 41 mothers mentioned whether or not the child was wanted. Of these, only 16, or 39 per

*CLEMENS E. BENDA. *Mongolism and Cretinism.* Grune and Stratton, New York. 1946. 298 pp. 12 pp. index. 103 figures. 48 tables. $6.50.

cent, wanted the child who later turned out to be a mongoloid. Again his generalizations do not fit his figures.

"Hypothyroidism . . . is frequently observed in mothers of mongoloids." (p. 287.) But in Table 43, Benda shows that it was observed in only 10 mothers, or 13.3 per cent. Again this was from the selected group of 75 mothers who showed at least one of his "constellation of factors."

Misconceptions Concerning Heredity

The arguments with respect to heredity are no more valid than the generalizations quoted above. Here is an example. "According to mendelian expectation, the incidence of any hereditary condition should increase in proportion to the number of offspring." There are several misconceptions evident in this statement, most important being the misuse of the word "incidence," and the erroneous use of the word "proportion." With any pair of parents of a given genetic constitution, the *incidence* of an hereditary trait in the offspring is independent of family size, and is determined by the mode of inheritance of the genes involved. Thus two parents, heterozygous for a recessive trait, will have approximately 25 per cent of the offspring exhibiting the recessive trait, no matter whether they have two or 20 children. It is true that the *number* of children showing any trait, be it hereditary or environmental in origin, increases as the size of the sibship increases. This is obvious, inasmuch as it is impossible for a family of two children to have more than two affected with a trait, whereas a family of 12 can show as many as 12 affected. This, however, is not determined by any "mendelian expectation," it is inherent in the fundamental axioms of mathematics, such as "The whole cannot be greater than the sum of the parts."

It is also true that the *percentage* of affected children in such families *falls* as the size of the sibship increases, *if one regards only those families in which the trait has been expressed* in at least one child. It is 100 per cent in affected progenies of one child, and falls to 57 per cent in progenies of two, and approaches 25 per cent as a limit, reaching a value of a little more than 25 by the time the sibship size has reached 12. If Dr. Benda means by "incidence" the ratio of affected to normal in total offspring of all heterozygous parents, he is wrong since this remains *stationary*. If he means the ratio of affected to normal in the offspring of those heterozygous parents who chance to have affected offspring, again he is wrong since this *decreases* with increase in size of sibship. If he means by the word "incidence" actual number of affected children, "mendelian expectation" plays no part. His thinking on this point is, to put it generously, confused.

Finally Benda states that the incidence increases in *proportion* to the number of the offspring. Proportion is a mathematical concept, and when one says that one value increases in proportion to the increase in a second, one expects that the first value always maintains a definite relationship to the second, not merely growing larger as the other increases, but growing larger in a definite ratio. This is not true of the only value which does increase as the number of offspring increases, namely, the *number* of affected children.

He states that in 255 cases of mongolism there were only two "incidences" (sic) of multiple mongolism, and that this does not prove inheritance. On the other hand he states 253 families with one to 15 children and with but one mongoloid "clearly demonstrate the absence of hereditary factors." This is not necessarily true. The literature of genetics contains many instances of two or more independently inherited recessive factors acting to produce a trait. In such situations the frequency of affected offspring is much less than when only one factor is involved. The larger the number of factors, the fewer will be the families with more than one affected

child. Even when only one recessive gene conditions a defect and both parents are heterozygous for it, we find more sibships with only one child affected with the defect than we find sibships with more than one affected. This is because most human sibships are small. When the sibship consists of five or less, a random group of families will have a majority showing only one affected offspring. It is not until the sibship size increases to six that one would expect more instances of two or more children affected than of one affected. Most families have less than five children. Dr. Benda's Table 39 shows that in his series of 253 mongoloid families, 180 (or 70 per sent) had less than six children. Therefore in 70 per cent one would expect to find more isolated examples of mongolism than of sibships showing a "familial" distribution. This is true even of conditions in which the expression of a defect is dependent upon only a single recessive gene. The small number of families which have more than one child affected suggests that mongolism depends for its expression on more than one gene, if the disease has an hereditary basis. By no stretch of either the imagination or of the rules of proof does this low incidence "clearly demonstrate the absence of hereditary factors."

We read on page 249 that "The incidence of hereditary anomalies is likely to increase if consanguinity is present in the parents." It is obvious that if both parents are heterozygous for a recessive trait, the fact that they are related does not increase the probability that their offspring will be affected. The expectation is identical in the offspring of unrelated and of related parents. If the trait is a dominant one, the fact that the parents are related does not increase the probability of the children being affected. What the author probably means is that the rate of consanguinity among the parents of offspring affected with a rare hereditary unit recessive trait is higher than the consanguinity rate among the general population. The rarer the trait, the higher the consanguinity rate in the parents of the affected offspring. If the trait in question is dependent upon more than one recessive trait, the excess of consanguineous marriages among the parents of affected offspring is much less noticeable.

Confused Thinking About Twins

When Benda comes to the analysis of twins (p. 249) he is even more confused. He says that of 100 twins (he means *pairs* of twins) 70 may be expected to be of the same sex and 30 of the opposite sex; and that of the 70 pairs of the same sex, 50 pairs are supposed to be dizygotic, and 20 pairs monozygotic. First, most authorities state that twins in general occur in the ratio of one pair of male twins to one pair of female to one pair of twins of both sexes. In 300 pairs of twins 100 pairs would be both males, 100 pairs both females, 100 pairs male and female. Dizygous twins occur in the ratio of one pair of males to one pair of females to two pairs of male and female twins. Therefore, there must be 50 pairs of dizygous male twins and 50 pairs of dizygous female twins to match the 100 pairs of dizygous twins of opposite sex mentioned above. This leaves 50 pairs of monozygous male twins and 50 pairs of monozygous female twins to make up the 300 pairs mentioned. In other words, one-third *of all twins* are dizygous and of opposite sex. Of the remaining two-thirds of all twins, one half of them are likely to be dizygous twin pairs of the same sex, and the other half are monozygous twins who are, of course, of the same sex. The following table shows the correct percentages of twins of different types.

Percentages of twins of different types.

	Monozygous	*Dizygous of same sex*	*Dizygous of opposite sex*
Correct estimate	33.3	33.3	33.3
Benda's estimate	20.0	50.0	30.0

After having made this false start, Dr. Benda shows further confusion on the subject of concordance and discordance in twins. If a trait is hereditary, both monozygous twins will show it if one of them does. On the other hand, one dizygous twin *can* show an hereditary trait

without the other showing it. The degree to which monozygous twins and dizygous twins differ in concordance depends on at least two factors, (1) the genetic makeup of the parents, and (2) the complexity of the mechanism which is responsible for the trait. If both parents are blood group O, for example, all their children, whether they be single births, dizygous or monozygous twins, will be blood group O. Dizygous twins will then show as much concordance in this respect as do monozygous twins. This does not preclude the genetic basis of blood groups, however. With respect to the second factor mentioned above, it is obvious that if one parent is heterozygous for a dominant trait of polydactyly, dizygous twins have an even chance of being concordant if one of them is affected. They will show concordance half as often as will monozygous twins. But if the trait is one dependent upon the simultaneous presence of *two pairs* of recessive genes for which both parents are heterozygous, then the chances of both being affected if one is are only one in 16, so that now the monozygous twins show concordance 16 times as often as the dizygous. The mere fact that dizygous twins are both affected with a trait does not show that it is not hereditary.

Hence Benda's statement that four instances of mongolism in twins "which were undoubtedly dizygotic is the most important observation of recent years" leaves the reviewers puzzled as to why he thinks it so important. He does not deign to enlighten us. But he is completely wrong in his inference that this observation destroys the last vestige of proof that genetic factors are important. Assuming that the twins were "undoubtedly dizygotic," we are not surprised by their both being affected; it is no more surprising than familial occurrence of mongolism, when two children in one family, who were not born as twins show the condition. Yet Benda would not maintain that familial instances of mongolism argue against heredity! Dizygous twins are merely siblings who are born at the same time. One swallow does not make a summer, nor do four pairs of mongoloid dizygotic twins annihilate heredity; in fact, they merely add more familial examples. Unfortunately, Benda has missed the significance of the twin data on both counts. He feels that mongoloid concordance in dizygous twins disproves heredity which it does not; and he fails to see that the large number of discordant dizygous twins is the strongest argument against the thesis that maternal, pre-natal conditions cause mongolism.

Benda explains mongolism as the result of deficient or unbalanced pituitary hormones in the mother during pregnancy. If a trait is determined by pre-natal factors not genetic in origin, both members of a dizygotic twin pair should show the defect if one does with the same consistency that both members of a monozygotic twin pair show it if one does. The majority of dizygotic twin pairs in which mongolism is present show only one twin affected, whereas *all* monozygotic twin pairs show both affected if the condition is present at all. Benda attempts to explain this discordance in dizygous twins by saying that when one twin only is affected, it is because the deficiency in the mother was not great, and only one twin was affected. The same blood, exhibiting the same deficiency in hormones circulates through the placentas of both dizygous twins; if they select differentially because of different thresholds of response to the hormone deficiency, the logical conclusion is that the difference was caused by a different hereditary makeup. It is incumbent upon Benda to explain why, when the twins are monozygotic, the maternal deficiency is always great enough to affect both twins; and why, when they are dizygotic, the deficiency is almost always so small as only to affect one. One has to understand the implications of the evidence offered by twin studies before one can draw valid conclusions.

Advice for Mothers of Mongoloids

Another evidence of confused thinking is his statement that a mother who

has produced one mongoloid child can be encouraged to have more children without risk. He adds, however, in the next sentence that it is advisable for a physician to study the *family background* because some instances are known in which such a misfortune has occurred twice. Why study the family background if heredity plays no role? Also, if a woman of 40 has one chance in 25 of producing a mongoloid child, and if her chances rise to one in eight when she is 45, such an attitude of encouragement is not justified.

This review is not intended to state that hypopituitarism in the mother or in the fetus plays no role in the causation of mongolism; nor that maternal age is not also a significant factor. It merely points out that Benda does not follow logical processes in analyzing his data; that he gets an impression from a few cases and enthusiastically sweeps "most" of the cases into the same category.

Case-History Pitfalls

He writes up histories in which the mother "felt ill" or "wept," or "did not feel the same as in her other pregnancies," and naively concludes that "no one who glances through the series of case histories above can escape the conclusion that the cause of mongolism is to be found in the condition of the mother at the beginning of pregnancy." He gives histories of mongoloid children being produced by women who felt ill or depressed or nauseated; by women who did not want to be pregnant or who wanted the child; by women who showed too much or too little thyroid; by women who had irregular menstruation before pregnancy, or bleeding during pregnancy; by women who merely threatened to have, or who actually did have abortions; by women who had hordes of children before they were 35, or by those who had but one when they were 45. We could match every one of these by a history of a woman with a similar experience, who produced a normal child at the end of the pregnancy. Even if these histories revealed a significantly higher percentage of mothers of mongoloids, we must remember that when a child is abnormal the mother will recall every little ill in order to explain it, whereas if the child is normal, she will forget these, not being stimulated to remember that which produced no visible effect. The only way in which an accurate evaluation can be obtained of the role played by these factors in causing mongolism is to take the histories of a great many women of varying ages *while they are pregnant,* before the birth of the child. If then one finds these conditions existing in a significantly higher percentage of mothers who later produce mongols as contrasted with those mothers who produce normal children, some reliance can be placed upon the validity of the fact of their occurrence, even though the validity of interpretation of the fact may be called into question.

The writing tends often to be careless, as when a child is described as dying of placenta previa, when of course it was the mother who had the placenta previa, although it may have been the child who died. "Incidence" was used in place of "instance." Some of the unfortunate misuses of language which have crept into medical terminology are found, such as "the mother was operated for tumor"; "pathology" for pathological lesions. He says that "Bad health during pregnancy was no significant item." Did he mean "insignificant"? In some places, he shows a complete misunderstanding of the processes involved. Thus he tests the *mother* of the mongoloid child for phenylpyruvic acid, when of course it is the feebleminded person who is the excretor of this acid, not the parents.

The scepticism engendered by the above considerations should not blind us to the possible importance of one observation which Benda makes, and which we have seen reported in no other study of mongolism. He states that women who produce mongoloid children have their normal children at an average age greater than that at which the control mothers were producing children of simi-

lar birth rank. He does not give in detail the figures on which this statement is based. From the data published in the book it is impossible to estimate the ages at which the mothers producing control and mongoloid children were having their first, second, etc., normal children. It would be very important to have this information published. It is evident from Benda's figures that mongoloid-bearing mothers have a reduced fertility, first in that they produced only about 75 per cent as many children as the mothers of the controls; second, that the period of reduced fertility seems to be most marked in the younger ages. Certainly mothers who produced their mongoloid children before the age of 35 are less fertile than the controls. Unfortunately, the manner in which the data are recorded does not permit of accurate determination by the reviewers of the extent of these differences. We hope that Dr. Benda will publish the details of the obstetrical history of these mothers, both control and mongoloid-bearing, giving the data on the date of birth of each child in the family, the date of abortions, and the length of the pregnancy in each case, the date of the mother's birth, the date of marriage, whether childlessness was voluntary, etc., so that those interested can compute from the original data the average age of the mothers at the birth of children in any specific birth rank, the intervals between births, etc. Such information may give us valuable clues to the maternal factors which may be present before a mongol child is born.

MADGE T. MACKLIN
LAURENCE H. SNYDER
Ohio State University

Blindness and Heredity

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among nearly 70 per cent of the pupils enrolled in schools and classes for the blind during 1943-44. These figures show that *55 per cent of the cases originated before birth.* Only 13 per cent, however, could be established as *hereditary in origin—meaning that the eye defect was present from the moment of conception.* The causes of the others were not specified, but many, undoubtedly, were congenital: The child was born with the defect but it wasn't present in the germ plasm itself; nature, for reasons unknown, had done a bad job.

That hereditary defects must necessarily be "present from the moment of conception" represents a novel form of a popular delusion. At conception the new individual consists of a single cell, lacking just about all the attributes which will characterize him as an adult —among them, eyes. Obviously, defects scheduled for later manifestation cannot be present at conception, nor need they be present at birth. Many hereditary characteristics do not manifest themselves until after middle life,—longevity for example. Hence the concept that heredity must be expressed at birth is completely erroneous. The author seems to have achieved in new high in confusion and mixed thinking in his differentiation of "present at conception" defects as "hereditary" which represent only a quarter of the congenitally blind. How these seemingly mutually exclusive concepts are to be reconciled is left to the reader.

The confusion resulting from this misunderstanding detracts greatly from the value of this section of the booklet. Actually the reader cannot determine how the author arrived at the estimate of 13 per cent hereditary blindness. Glaucoma is not present at birth and does not generally appear until late in life, and in many families is definitely an hereditary disease. Juvenile cataract is usually present at birth but it may be manifested

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COLOR ON THE LEGS OF SHEEP

Its Inheritance in the Columbia and Targhee Breeds

CLAIR E. TERRILL
*United States Department of Agriculture**

BROWN or black color or spots may occur in the hair on the lower part of the legs in many breeds of sheep. The same kind of color variation is often found on the face. Most associations for breeds of white-faced sheep consider this color with disfavor. Brown color is usually considered less acceptable than black, although these colors often occur together in various gradations. The basis for discrimination against color on the legs in selecting breeding sheep has not been clearly defined. Leg color in white-faced breeds may give an undesirable appearance and also detracts from uniformity of appearance. However, there appears to be no association between the absence or presence of color and mutton conformation, quantity or quality of wool, or lamb production. Leg color may be considered in selection partly because it is more easily recognized than are differences in lamb and wool production.

In 1936-37 observations were made of color on the legs of all sheep in the U. S. Sheep Experiment Station flock. In addition, preliminary matings were made of a ram with leg color to ewes both with and without color. The proportion of offspring with color from these matings was even less than for the remainder of the flock, in which no rams with color were used. The ewes with color when mated to the ram with color had only slightly more offspring with color than ewes without color. These results indicated that the inheritance of leg color is complex and that the culling of sheep with color will result in only a slight reduction in the proportion of offspring with color in the flock. Consequently it was tentatively decided to reduce emphasis on leg color in selection of ewes but to continue to record routine observations on the occurrence of color so that a more complete analysis could be made later.

The object of this study was to obtain information on the occurrence, inheritance, and relation to other traits, of leg color in order to determine the relative emphasis to give it in a breeding program for improvement of range sheep.

Material and Methods

Data were used from 1,517 Columbia and 1,051 Targhee weanling lambs born from 1941 to 1945. These scores were taken at weaning time in the latter part of August when the average age of the lambs was 120 days. Scores for leg color from their sires and dams as weanlings were also used. In a few cases, color scores for sires and dams were taken later than weaning age. The degree of brown or black color on the lower part of the legs was scored from 1 to 5 as follows:

1—white with no colored spots or fibers; 2—slight color with a few brown or black spots or fibers; 3—a medium degree of color; 4—considerable color; 5—a solid color or colored spots covering the legs.

Only data for lambs born from 1943 to 1945 were used in determining the effect of environmental factors on leg color and the relation of leg color to other traits. Environmental factors studied included year, sex, twinning, and age of dam. Other traits studied in relation to leg color included face covering, length of staple at the side, body weight, type, condition, and neck folds.

Estimates of heritability were obtained from half-sib correlations and regressions of offspring on dam which were calculated for each breed each year. The half-sib correlation was multiplied by four and the offspring-dam regression by two to obtain estimates of heritabil-

*U. S. Sheep Experiment Station, Dubois, Idaho.

ity, since the relationship between half-sibs is 0.25 and that between dams and offspring is 0.50 in non-inbred populations. Inbred lines of 30 to 45 ewes each were started in these breed groups in 1941 but the slight inbreeding from this source appeared to have been offset by the relationship between lines due to common inheritance from foundation animals. Standard errors for the half-sib correlations and offspring-dam regressions were calculated as shown by Hazel and Terrill,* these being multiplied by four and two, respectively, to obtain standard errors for the individual estimates of heritability. Estimates of heritability were averaged by weighting each estimate according to the reciprocal of its squared standard error.

Results

The proportions of offspring and dams with various degrees of leg color are shown in Table I. About 12 percent of Columbia offspring and 15 percent of Targhee offspring had leg color. The percentage of dams with color was higher in each breed than for the offspring. This indicates that some progress has been made in eliminating color through selection of the sires. Most of the rams selected for breeding were free of leg color and had color scores of 1. Only a few rams with slight leg color were used as four of the Columbia and five of the Targhee sires had color scores of 2. Some selection against extreme color in ewes was practiced as only two of the dams had color scores of 5 as compared with 18 offspring. No relation was found between leg color and other weanling traits including face covering, length of staple, body weight, type, condition, and neck folds. The occurrence of leg color was unaffected by sex, twinning or age of dam. Highly significant differences between years were found in the occurrence of leg color in weanling lambs. Yearly changes in proportion of lambs with color were consistent for the two breeds. The highest incidence of color occurred in 1942 and the least in 1944. These yearly variations are difficult to explain. Misclassification might cause such differences but all observations were made by the same worker. There were yearly differences in the amount of stain and dirt on the legs which made the scoring of color difficult but no records were kept of such conditions. Chance variation in the genetic makeup of the sires might cause some yearly change.

Some information on the inheritance of leg color may be obtained from the matings made in the regular breeding program, although they were not specifically planned for this purpose. Parents without leg color when mated together gave 7 percent of Columbia and

TABLE I.—Occurrence of leg color in Columbia and Targhee flocks

Breed	Year	No. of Sires	No. of offspring—dam pairs	Percent of offspring with score of 1	2	3	4	5	Average color score of offspring	Percent of dams with color score of 1	2	3	4	5	Average color score of dams
Columbia	1941	10	216	85	6	6	3		1.28	74	14	9	3	..	1.42
	1942	11	264	84	6	6	3	1	1.33	78	13	8	1	..	1.33
	1943	10	261	94	3	2	..	1	1.11	78	13	9	..	..	1.31
	1944	12	355	94	3	3	..	..	1.09	80	13	7	..	..	1.27
	1945	12	421	85	8	4	3	..	1.27	84	9	7	..	..	1.24
All years		55	1517	88	5	4	2	1	1.21	79	12	8	1	..	1.30
Targhee	1941	8	150	83	7	9	1	..	1.29	78	18	3	1	..	1.28
	1942	10	217	75	11	9	2	3	1.47	76	17	5	2	..	1.33
	1943	9	194	83	8	7	1	1	1.28	79	13	5	3	..	1.31
	1944	8	221	91	5	3	..	1	1.15	77	17	4	2	..	1.30
	1945	10	260	89	5	4	2	..	1.19	84	10	4	2	..	1.25
All years		45	1051	85	7	6	1	1	1.27	80	14	4	2	..	1.29

*Hazel, L. N. and Clair E. Terrill. *Jour. of An. Sci.* 4(4):347-358. 1945.

12 percent of Targhee offspring with leg color. The respective percentages were 22 and 24 when one parent had leg color and 44 and 38 when both parents had leg color. In general the proportion of offspring with leg color increased with an increase of degree of leg color in the parents. Mating of rams without color to ewes with 2, 3, and 4 color scores resulted in 16, 19, and 50 percent of offspring respectively with leg color in Columbias and 16, 42, and 29 percent in Targhees. Mating of rams with a color score of 2 to ewes without color resulted in 32 and 25 percent of Columbia and Targhee offspring respectively with color. These percentages were higher than in the corresponding reciprocal cross, in which 16 percent of offspring with color were produced in each breed.

Discussion

The above data on the inheritance of leg color do not lead to a simple genetic interpretation. A considerable number of lambs with color are born from parents without color, which may indicate that leg color is recessive. However, when both parents had leg color less than half of their offspring had leg color. This indicates that more than one pair of genes are involved. The increasing proportion of offspring with color as the degree of color increases in the parents indicates that the genes involved are cumulative in their effect.

The results may have been over-simplified because all degrees and kinds of color on the legs have been recorded within the narrow range of only five classifications. Added refinement and detail of describing color on the legs might have led to more simple and complete genetic explanations. However, further refinement in measurement of leg color on range sheep was not practical in view of the questionable economic importance of the trait.

As already stated, heritability of leg color was estimated from intra-sire regressions of offspring on dam and from half-sib correlations. The latter gave lower estimates probably because the sires had been selected for absence of color. For this reason they are probably less reliable than estimates from the other method. Estimates of heritability from intra-sire regressions of offspring on dam were 0.26 ± 0.05 and 0.34 ± 0.07 for Columbias and Targhees respectively. In general, these estimates are consistent with progress made from selection against color in rams. Sires averaged about 0.2 score better than dams. This selection differential when multiplied by a heritability of 0.3 and divided by 2, because the selection was confined to sires, would indicate a probable gain of 0.03 score per generation. The average gains of the daughters over the dams for the period studied were 0.09 and 0.02 scores for Columbias and Targhees respectively.

Further improvement in elimination of leg color in these breeds is certain to be slow. Complete culling against color would still leave about 7 percent of Columbia and 12 percent of Targhee offspring with leg color. The respective percentage of color would be 12 and 15 percent if rams only were culled for leg color and slightly more than 20 and 21 percent if there was no selection for absence of color.

Complete selection against leg color would give this trait about equal emphasis in selection to other weanling traits for ewe lambs and lower emphasis for ram lambs. That is, the proportion of ewe offspring culled for color would be similar to the proportion culled for short staple, small size, or poor type. Certainly, little is to be gained by selecting against leg color in ewes. Selection against color in rams attaches an economic importance to the trait which is difficult to justify because of the lack of relationship of leg color to market value of lamb or wool.

Summary

The inheritance of brown or black color on the lower part of the legs was studied on 1,517 Columbia and 1,051 Targhee weanling lambs born from 1941

to 1945, and on their sires and dams. Twelve percent of Columbia lambs and 15 percent of Targhee lambs had leg color. The percentage of dams with color was higher in each breed. This indicates that little selection against color has been practiced in ewes but that some progress has been made in eliminating color through selection of the sires.

No relation was found between leg color and other weanling traits including face covering, length of staple, body weight, type, condition, and neck folds. The occurrence of leg color was unaffected by sex, twinning or ages of dam.

Leg color appears to be recessive because many lambs with color are from parents without color. However, when both parents had color less than half of the offspring had color indicating that more than one pair of genes are involved. The proportion of offspring with color increased as the degree of color in the parents increased indicating that the genes involved are cumulative in their effect.

Heritability of leg color was estimated from intra-sire regression of offspring on dam and from half-sib correlations. The latter gave lower estimates probably because the sires had been selected for absence of color. Estimates of heritability from intra-sire regressions of offspring on dam were 0.26 ± 0.05 and 0.34 ± 0.07 for Columbias and Targhees respectively. These estimates are fairly consistent with progress made from selection. Further progress toward eliminating leg color will be slow because its incidence is already low. Culling of all sheep with color would reduce the percentage of lambs with color to about 7 in Columbias and 12 in Targhees.

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Blindness and Heredity

(*Continued from page* 88)

later. Yet the author recognizes it as definitely hereditary. Surely more than one in eight of those born blind must owe their condition to genetic factors. The author rightly recognizes that persons who are blind or have blind relatives have a definite responsibility to determine whether this affliction is genetic. The means suggested to determine this are, unfortunately, wholly inadequate: "The answer should come from an enlightened doctor who has studied the patient and knows the family's record. It is still hard to differentiate between hereditary and congenital causes, but if there have been no other cases of serious eye trouble in the family for several generations, the chances are that the difficulty will not be passed on."

Since most doctors are wholly unacquainted with even simple genetic principles, those who follow this advice may have some unpleasant surprises in store. Cases are on record where people have asked for advice from a doctor who knew the family history. "Not genetic" was the verdict in one case, and it was followed by the birth of five blind children. Many doctors are almost as badly confused as the author of this booklet concerning the genetic implications of "congenital" and "familial" traits. There are a multitude of instances in which recessive defects have cropped out when the ancestry of neither parent had any record of the trait. Albinism may be cited as an example. It is "congenital" and it frequently appears among the offspring of normal parents whose ancestors showed no albinism. Nevertheless it is definitely hereditary, and two such "congenital" albinos, if they marry, will have no normal children. Recessive blindness acts in exactly the same way.

The author is on the right track in emphasizing that heredity is an important factor in the problem of blindness even though he underestimates it. It is tragic, however, that an otherwise excellent handbook should be marred by these serious misapprehensions and dangerous misstatements regarding the nature of genetic processes.—R. C.

THE INHERITANCE OF STREBLOMICRODACTYLY

LAURENCE M. ASHLEY
College of Medical Evangelists, Loma Linda, California

CROOKED LITTLE FINGERS
Figure 12

The inability to straighten the little finger is a minor defect, of no great importance to the individual. It prevents normal manipulation of the fingers in such coordinated fine motion as piano playing.

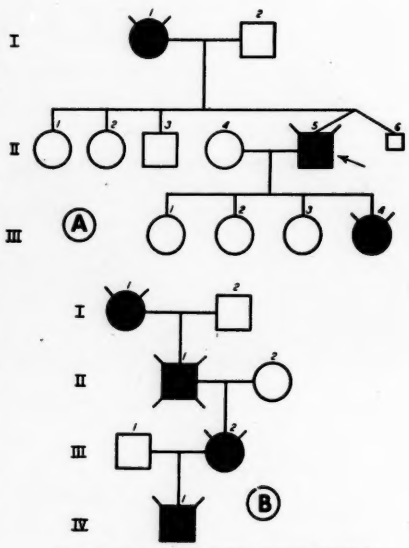
STREBLOMICRODACTYLY is the term proposed by Heffner[1] to describe that congenital anomaly caused by a short tendon of the flexor digitorum sublimis muscle in either or both of the little fingers. The condition is hereditary and until recently has been thought to be transmitted as a simple dominant. It now appears that its mode of transmission is not always simple but that the influence of one or more genetic modifiers may result in variable expression of the dominant gene. In a more recent paper, Hefner[2] described a second type which is characterized by a very slight degree of flexion of the little finger. This form of the defect he termed "minor" streblomicrodactyly. Hefner suggested the possibility of the major and minor forms being conditioned by multiple alleles but admitted that in the absence of pedigrees exhibiting both types or "degrees" of flexion his hypothesis was purely conjectural. That Hefner's two hypothetical forms of the defect may occur in the same individual and in different individuals in the same family is evident from a study of the pedigrees presented below. More recently, Spear[4] hypothesizes the interaction of one or more modifying genes, with the generally accepted dominant gene for the various occasional flexions of digits other than the little fingers. Further variability has been noted by Moore and Messina[3] to the effect that many persons so affected exhibit finger flexion in only one hand. This, they assume, indicates that the expression of the defect is strongly influenced by environmental factors. Stoddard's[5] observation that early repeated massaging of the shortened flexor tendons seems to ameliorate the condition testifies to the effects of such therapeutic measures.

Pedigree A

Figure 13, upper, covers three generations of streblomicrodactyly. It is expressed in the major* form in the mother (I-1) and in one of her five children (II-5), whose twin brother (II-6) died in infancy and nothing is known concerning his fingers. One of the four daughters (III-4) of the propositus (the surviv-

*These diagnoses as to form are based on examination by the author, except for that of I-1 which is based on the testimony of II-5 whose reliability I will vouch for.

ing twin), has minor streblomicrodactyly. The condition is not noticeable, her little fingers appearing to be straight, except that upon close scrutiny they show slight evidence of contraction. Each of the affected persons in this pedigree manifests bilateral involvement which is approximately equal in degree on the two sides. The severity of the flexion of his mother's little fingers is attested to by the propositus who writes: "I remember mother remarking that as a young girl she suffered some inconvenience when it came to wearing gloves and in practicing on the piano. She used to hammer the fingers with a wooden mallet in order to try and stretch them." He says of himself: "My most serious inconvenience was in school in the old days when we used to get severly caned on the hands, and my little fingers used to get more than their share of the punishment until I learned to hook them underneath the third (ring) fingers."



CROOKED FINGER PEDIGREES

Figure 13

The symbols are explained in the chart shown in Figure 14. Individual II-I in Pedigree *B* is unique in having both fingers and toes affected.

Pedigree B

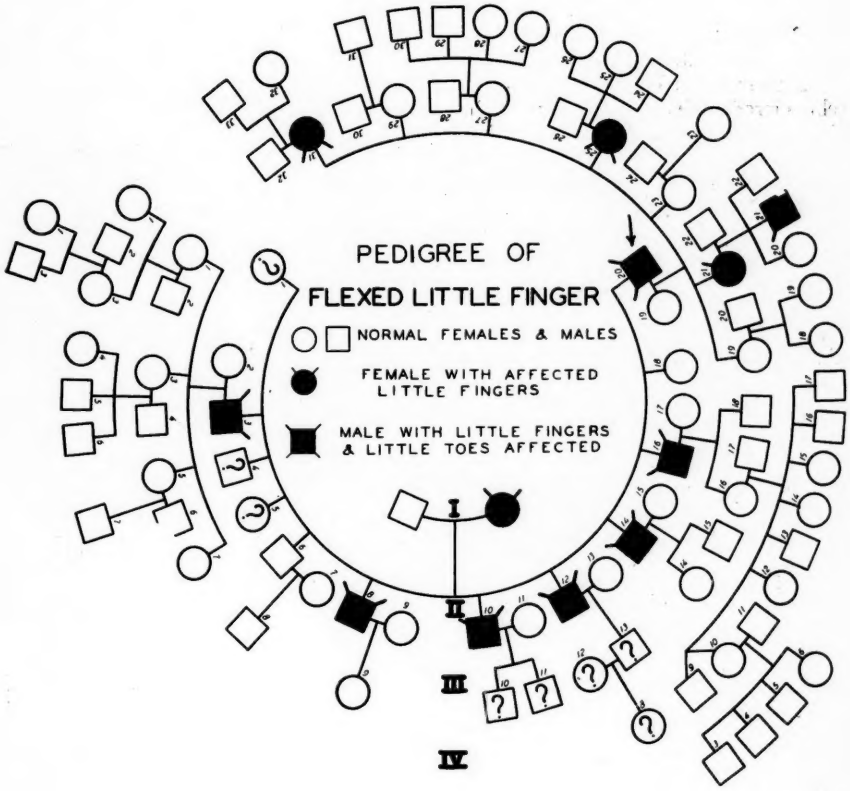
The *B* family shows the trait in only a single sibling in each of four generations. (Figure 13, lower.) The mother, I-1, had brothers and sisters but nothing is known concerning them or their parents. Her toes were normal but her only child (II-1) had flexed little toes as well as flexed little fingers. His daughter (III-2), also an only child, has bilateral little finger flexion which is more severe than that of her father but her toes are all normal. Her only child (IV-1) has both little fingers affected and his toes are normal. His fingers are less severely flexed than are those of his mother.

Pedigree C

In this family, which covers five generations, streblomicrodactyly is bilateral in all affected members of the first and second generations but this is true in only half of the affected members of the third and fourth generations. None of the fifth generation are known to be affected. In the second generation seven of the nine siblings for whom information is available are affected. (See Figure 14.) With the exception of II-20, the progeny of this sibship (numbering three generations in two cases) all lack the defect. This may be misleading, since nothing is known of the offspring of two of the brothers (II-10, 12).

The almost complete lack of the anomaly in the third, fourth and fifth generations is believed to be due to successive diminution in degree of penetrance probably brought about through the introduction of modifying factors inherent in the unaffected spouses of the affected second generation siblings.

II-3 had major flexion of the right little finger but only minor flexion of the left. Nothing is known of the fingers of II-1, 4, 5 who died of diphtheria in early childhood. The condition of the



DOMINANT INHERITANCE

Figure 14

In the first few generations the affected fingers are on both hands. Two of those in the late generations are unilaterally affected, suggesting a dilution effect.

propositus (II-20) is similar to that of II-3, except that as a result of repeated massaging, the right little finger is definitely less flexed now than formerly. This bears out Stoddard's observation, noted above.

Although not shown as affected in Figure 14, III-19, and her daughter, IV-19, each have clinodactyly of the little fingers, of the type described by Zurukzoglu[7] in which the affected digits are bent radially or towards the thumb. Similarly affected are IV-29 and IV-30 although their mother is normal. It is possible that these four clinodactyls should be included as affected members of the pedigree even though their anomaly is of a different, although similar, type. It is conceivable that the same gene, under the influence of either genetic or environmental modifiers (possibly intrauterine), may be responsible for both types of finger bending. Of the seven flex-fingered males in the second

generation, one of them, (II-16), also had clinodactyly* of his little fingers.

The typically affected progeny of the propositus include: III-21, who exhibits streblomicrodactyly only in her right little finger; her son (IV-21) who has the anomaly only in his left little finger; III-25, whose right finger is affected and whose left finger shows a mere trace of flexion; and III-31 (Figure 12) who has a well flexed left little finger but only minor flexion in the right one.

The Three Pedigrees Compared

Analysis of the three pedigrees (Figures 13 and 14) reveals a remarkable similarity between them in that inheritance in each is from mother to son and from father to daughter. This resemblance to sex-linked inheritance is, however, probably due to coincidence inasmuch as the pedigrees exhibit too many affected females for ordinary sex-linked recessive inheritance and too few affected females for dominant sex-linkage (rare in man). This coincidence appears to be largely dependent upon the fact that the sex ratios in these families tend to be predominantly male in one generation and female in the next, etc. It is, therefore, concluded that the pedigrees here presented are examples of irregular dominant inheritance which is the usual type for streblomicrodactyly. There is considerable variability of expression and the average degree of penetrance for the three cases combined is only thirty-two percent (thirty-nine percent, if the four "clinomicrodactyls" mentioned above are counted along with the sixteen "camptomicrodactyls.") This is based upon a total of 101 offspring, excluding the first generation, and omitting persons for whom information is lacking.

These pedigrees offer no clear cut evidence in favor of linkage between the finger defect and hair color, as reported by Tomesku[6] and Spear.[4]

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*The writer would like to suggest, for the sake of brevity and systematic nomenclature, that if we are going to persist in using Hefner's term, "streblomicrodactyly" for flexed little fingers, we should adopt the term "clinomicrodactyly" for little fingers bent radially (lateral in anatomical position). Some authors may prefer the term "camptomicrodactyly" to streblomicrodactyly since the former derives from "camptodactyly," a term evidently holding priority over the synonym, "streblodactyly."

◆

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